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A new hope for obesity management: Boron inhibits adipogenesis in progenitor cells through the Wnt/β-catenin pathway

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ABSTRACT

Obesity is a worldwide medical problem resulting in serious morbidity and mortality involving differentiation of pre-adipocytes into mature adipocytes (adipogenesis). Boron treatment has been reported to be associated with weight reduction in experimental animals; however, its effects on pre-adipocyte differentiation and anti-adipogenic molecular mechanisms are unknown.

In this study, we demonstrate the inhibitory activities of boric acid (BA) and sodium pentaborate pentahydrate (NaB) on adipogenesis using common cellular models. Boron treatment repressed the expression of adipogenesis-related genes and proteins, including CCAAT-enhancer-binding protein α and peroxisome proliferator-activated receptor γ, by regulating critical growth factors and the β-catenin, AKT, and extracellular signal-regulated kinase signaling pathways. In addition, although boron treatment did not induce apoptosis in pre-adipocytes, it depressed mitotic clonal expansion by regulation of cell cycle genes.

Overall, these data offer promising insights into the prevention/treatment of obesity and associated diseases.

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1. Introduction

Obesity and associated comorbidities, such as type 2 diabetes, hypertension, cardiovascular disease, cancer, and metabolic syndrome or posttraumatic stress disorder (PTSD), are worldwide health concerns, particularly in developed countries, leading to increased healthcare costs, morbidity, and mortality [1,2]. An estimated 2.1 billion people worldwide are thought to be overweight or obese, and 2.8 million deaths are associated with obesity annually [3]. Although the exact molecular mechanisms of obesity are not well understood, uncontrolled hyperplasia and hypertrophy of adipocytes, the main units in fat tissue, are

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